# REMARKS ON GASTROSUCCORRHŒA AND TETANIC ATTACKS OCCURRING WITH CHRONIC ULCER OF THE STOMACH.[1]

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Ever since analyses of gastric contents have been made in order to study the function of the stomach and its disturbances mistakes have been made chiefly in two directions: certain functional disturbances which are really only symptoms have been stamped as diseases, and in explaining certain conditions too much stress has been laid upon secretory disturbances, the importance of motor disorders being underestimated. This applies also to the picture described as gastrosuccorrhœa. Although much has been written about this symptom-complex, there is still a great deal of confusion in regard to its correct interpretation, and it is therefore justifiable to consider once more whether this picture ought to be taken as an independent disease.

Reichmann was the first to apply the name "gastrosuccorrhœa" to a set of symptoms which are chiefly characterized by the presence in the fasting stomach of a fluid possessing all the chemical and physiological properties of gastric juice. The patients, who are generally emaciated, complain of pyrosis, eructation, and vomiting of highly acid matter, and suffer a great deal from gastric pains coming on after meals and during the night. Examinations of the stomach contents after test meals show hyperacidity and impaired digestion of starches. In the urine there is often a diminution, and sometimes even a total absence, of chlorides. In Reichmann's opinion the characteristic feature of this condition is the continual flow of gastric juice, not only after meals and during the process of digestion, but also during the intervals when the stomach of a normal individual should be empty.

Reichmann's publication aroused an extensive and lively discussion of the questions: Is gastrosuccorrhæa, as its name indicates, really a primary secretory disturbance, or is the accumulation of secretion in the fasting stomach the result of a motor insufficiency? Is gastrosuccorrhœa a disease *per se*, or is it merely a peculiar combination of symptoms which accompany other already well-known pathological conditions? A consensus of opinion on these questions has not as yet been reached.

There are two varieties of gastrosuccorrhœa—the periodic and the chronic. In the *periodic form* we have to deal with attacks of vomiting of gastric juice, coming on spasmodically and lasting from a few hours to several days. The condition has been attributed to a morbid overactivity of the secretory nerves of the stomach, which is regarded as a neurosis by itself, as a symptom accompanying hysteria or neurasthenia, or as a symptom occurring in the form of gastric crises in tabes and other spinal diseases. The remaining symptoms occurring with such attacks, pyrosis, headache, gastric pains, and vomiting, are classed as secondary manifestations. The same attacks have, however, been described by a number of other writers under different titles, the name depending upon the symptom regarded as being the most important. Thus, Leyden refers to them as "periodic vomiting;" Struebing classes them with disorders which he designates as "angioneurotic œdema;" Rossbach, who considers the increased secretion of hydrochloric acid as the chief factor in the condition, calls them "gastroxynsis." They are also found in descriptions of nervous diseases. Möbius, for example, associates them with migraine. I shall not further discuss this group of cases of periodic gastrosuccorrhœa, the pathogenesis of which is still very vague. The secretion of large quantities of gastric juice accumulating in the stomach independently of the ingestion of food may play an important role. However, all that has thus far been taught about the relation of the various symptoms to one another, and especially in regard to the point whether this increased secretion of gastric juice is the primary or only a secondary factor in the attacks, is still altogether hypothetical.

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In *chronic gastrosuccorrhœa* the conditions are entirely different, since it is not of a nervous character, but is dependent upon anatomical changes of the stomach or duodenum. In these cases it can be demonstrated that the presence of fluid in the fasting stomach invariably means a motor disturbance, and that the view that chronic gastrosuccorrhœa is a disease *per se* is not well supported.

As has already been mentioned, Reichmann's theory was that the characteristic feature of the disease was the presence of gastric juice in the stomach at a time when the organ should be empty. According to his view, this accumulation of gastric juice occurs because the mucosa of the stomach secretes continuously without any apparent irritation, but solely in consequence of the morbid activity of the secretory nerves of the stomach.

Reichmann, however, did not overlook the fact that the presence of gastric juice in the fasting stomach could not be explained simply as a hypersecretion. In some of the cases described by him as much as a litre of fluid rich in hydrochloric acid could be expressed in the morning. In these cases there was undoubtedly an increased secretion, but this is by no means the rule. In a number of other cases the quantity found was decidedly less. The amount of hydrochloric acid contained in the fluid also varies. In cases where only relatively small quantities are found, or where the amount of hydrochloric acid is low, we cannot very well speak of increased secretion, especially as we do not know the quantity secreted by the normal stomach during a given time. The essential point, therefore, is not that there is an increased secretion, but is rather in the fact that this secretion is found in the stomach at a time when the organ ought to be empty.

The opinion was formerly held that the fasting stomach of a healthy individual was entirely empty. This view has since been somewhat modified, Rosin and Schreiber having reported that in systematically examining a large number of healthy individuals they often found gastric juice in the fasting stomach in varying quantities, sometimes reaching 40 c.c. Martius, Hemmeter, and others have confirmed these statements. The gastric secretion in these cases is explained as being the result of the stimulation caused by swallowed saliva and pharyngeal mucus.

However that may be, quantities of 50 c.cm. and over are surely pathological. Where such quantities are found it is possible that gastric secretion has been increased and kept up beyond the time required for digestion. But the fact that it is found in the fasting stomach can only be explained by the existence of some hindrance to its passage into the intestine. For, if motor function of the organ is normal, there is no reason why such increased secretion should not pass into the intestine just as well as other stomach contents.

The misinterpretation of these conditions was partly due to the singular confusion which followed the discussion of the meaning of gastric dilatation. I shall not enter into the discussion of this subject further than is necessary for understanding its relation to gastrosuccorrhœa. There is no reason for discarding the old, well-defined clinical picture of gastric dilatation. Its characteristic feature is that it is a combination of two conditions which must be distinguished: the enlargement of the organ and the stagnation of stomach contents. Enlargement of the stomach is an anatomical condition. Stagnation of stomach contents represents a functional disturbance—a motor insufficiency. A stomach with normal motor activity, no matter what its size, empties its contents during the night. Where this is not accomplished motor insufficiency is present.

The stagnating material may consist principally of solid food remains mixed with a larger or smaller quantity of fluid, or it may be chiefly fluid containing few or no solid particles. When solid particles are found, all authorities agree on the presence of stagnation. But when only fluid can be expressed from the fasting stomach, as is often the case in gastrosuccorrhœa, some authors maintain that the condition cannot be considered a stagnation, because no solid particles are contained in the fluid. To support this view, it was argued that in some of these cases certain symptoms generally associated with gastric dilatation were absent. It was claimed, for instance, that in cases where no lowering of the greater curvature could be demonstrated there could be no dilatation of the organ, and therefore no motor insufficiency.

In diagnosing dilatation of the stomach we must separately consider three things: (1) the size of the stomach; (2) its position, and (3) its mechanical ability.

Size and position of the stomach do not necessarily indicate its mechanical ability. A stomach may be very large and its greater curvature may be low, yet it may be motorily sufficient. In such a case we cannot speak of dilatation, but simply of a deeply situated, enlarged stomach—megalogastria. On the other hand, a stomach may be high and its motility be insufficient. This very condition is not infrequently found with gastrosuccorrhœa. We shall see later on that in these cases we have to deal with gastric ulcer. When the ulcer is situated at the lesser curvature and there are adhesions to the surrounding tissues, the stomach remains high; and in case of dilatation extends upward. In these cases I have frequently seen the fundus of a stomach almost reach the axilla in the dorsal position. When the lesser curvature is thus fixed the greater curvature may remain above the umbilical line, even in cases of pronounced dilatation with decided stagnation of the stomach contents. Hence, a high position of the greater curvature cannot be regarded as proving that in a given case no gastric dilatation is present, and, therefore, no motor disturbance.

Just as invalid is the argument that in certain cases of gastrosuccorrhœa no motor insufficiency was present because certain forms of fermentation were absent, particularly the gas fermentation. It is true that whenever there is stagnation in the stomach fermentation sets in. There are, however, various fermentations in the stomach, brought about by different organisms and leading to different fermentation products. The absence of any one form does not indicate that all fermentation is absent. By careful analysis some form of fermentation can always be demonstrated.

As for the statement that a motor insufficiency cannot be present when the stomach contents expressed in the morning do not contain food particles, it must be said that actual findings do not justify this conclusion. Reports of autopsies in such cases, where only fluid could be obtained without food particles, showed pyloric obstruction with ulceration. Also, it must be remembered how difficult it is to empty the stomach entirely when dilatation is present.

We may add that in the majority of cases this fluid actually contains food particles. If we examine the histories of typical cases, as reported by Reichmann and others, we find it especially stated that the fluid expressed from the fasting stomach contained varying amounts of food particles, principally starches, and also sarcinæ, which, beyond doubt, indicate motor insufficiency.

From the foregoing statements, therefore, we see that all arguments brought forward to explain the presence of larger quantities of gastric juice in the fasting stomach merely as a result of secretory disturbance without motor insufficiency are invalid. We must insist that the accumulation of acid fluid in the fasting stomach is always the result of motor insufficiency. This motor insufficiency may be due to atony of the stomach or to organic or spastic stenosis at its outlet.

Let us now consider the question: Is chronic gastrosuccorrhœa a disease *per se?* The fact that the presence of gastric juice in the fasting stomach always means a retention of secretion due to motor insufficiency does away with one argument often put forth in support of the theory that gastrosuccorrhœa is a primary and independent secretory disorder—the argument that the secretion of gastric juice takes place without the influence of direct irritation (stimulation by food), but purely as the result of the pathologically increased activity of the secretory nerves. For even small quantities of food particles are sufficient stimuli for the secretion of gastric juice, particularly when they lie directly on an ulcer, and when nothing but fluid is retained this stagnating fluid acts as an irritant. The more sensitive the mucous membrane the greater the quantity which it will secrete. The constant irritation of this stagnating fermenting fluid must in time increase the irritability of the mucous membrane and lead to anatomical changes.

Examinations of the mucous membranes in these cases, made at first by Korzynski and Jaworski, and later by Hayem, Hemmeter, Strauss and others, show a definite form of gastritis characterized by a destruction of the chief cells, the parietal cells remaining intact. There was often found a proliferation even up to the formation of polypi.

In opposition to the view which considers the gastritis a result of the permanent irritation by the stagnating masses, due to the motor insufficiency, some authors claim that the gastritis is the primary element of the whole process, and assert that the inflammation of the mucosa alone suffices to cause an abnormal irritability and an increased and continuous secretion of gastric juice. Even the development of gastric ulcer is attributed to this gastritis.

Granting that the gastritis plays an important part here, it seems to me that there is no reason for dwelling on the question whether the motor insufficiency or the inflammation of the mucous membrane is the primary disturbance. Both are factors in producing gastrosuccorrhœa; in some of the cases the motor insufficiency being the primary trouble, in others the gastritis.

However gastrosuccorrhœa is brought on, since we have seen that gastric juice in the fasting stomach always means a motor insufficiency, there can be no doubt that the stagnating secretion keeps up and increases the inflammation of the mucous membrane.

It is clear from the above explanation that where motor insufficiency and increased secretion go together a retention of gastric juice can take place. Such simultaneous occurrence of motor and secretory disturbance is often found during the course of various diseases of the stomach. Thus the presence of the gastric juice in the fasting stomach is a condition frequently observed in gastritis acida with atony, atony with secondary gastritis and hypersecretion, pyloric and duodenal stenosis resulting from adhesions or compressions, etc. It is obvious that in all these diseases gastrosuccorrhœa can only be considered as a symptom.

Some authors apply the term gastrosuccorrhœa to all cases where gastric juice is found in the fasting stomach. Others maintain that we ought to distinguish between the cases where the presence of gastric juice in the fasting stomach is only a symptom, and another well-defined group of cases for which they would reserve the term gastrosuccorrhœa. They claim that the presence of gastric juice in the fasting stomach does not justify a diagnosis of gastrosuccorrhœa unless the completely developed symptom-complex of Reichmann is also present. They regard this symptom-complex (Reichmann's disease) as a sharply defined, typical picture, showing, besides the characteristic presence of hydrochloric acid in the residual fluid, a number of other symptoms—pyrosis, vomiting, loss of flesh, and, most important of all, of severe pains occurring after meals and during the night.

If we now scrutinize this limited group of cases, in which the symptom-complex of Reichmann is fully developed, it must be said that even for this group no proof has yet been given that gastrosuccorrhœa occurs as a disease *per se*. In fact, whenever an operation or an autopsy has afforded an opportunity to gain an anatomical basis it has revealed ulcer or its sequelæ. Thus we see that even in these cases, which apparently are so well defined, gastrosuccorrhœa is only an accompanying symptom of a well-known disease—*i. e.*, gastric ulcer.

As long as we have no proof that gastrosuccorrhœa may occur as an independent disease we shall do well to regard it merely as a symptom. In this smaller group of cases in which the combination of symptoms given by Reichmann is completely developed and where severe gastric pain plays an important role, we shall not err if we regard it in every case as a symptom of gastric ulcer. If we look upon gastrosuccorrhœa in this light as a symptom, it becomes a valuable aid in diagnosing certain cases of gastric ulcer. Whenever we find a patient suffering from gastric pains coming on regularly after meals and especially at night, with or without vomiting, the presence of acid fluid even in small quantities in the fasting stomach indicates ulcer of the stomach. Hemorrhage and perforation, the most characteristic symptoms of ulcer, are observed only in a certain percentage of the cases. In the remaining cases gastric ulcer may present many different clinical forms, among which

Reichmann's symptom-complex stands out as a very characteristic and typical picture.

As a rule, in cases which develop gastrosuccorrhœa the ulcer is situated near the pylorus and causes a mechanical obstruction, generally by spasm of the pylorus. This spasm, which brings on severe pain, usually occurs after the greater part of the stomach contents have passed into the intestines. That frequent spasms do really occur here is proved by the finding of a strongly hypertrophic pyloric ring.

In the treatment of gastrosuccorrhœa it is of great practical importance to have clearly in mind the fact that the motor disturbance plays a more important role than the secretory, and, further, that the well-developed symptom-complex of Reichmann is not a disease *per se*, but a symptom of gastric ulcer. We accordingly treat the condition by freeing the stomach from its stagnating contents, also using methods usually resorted to in gastric ulcer. Cures by such treatment have often been reported, and my own experience is corroborative.

In cases which do not yield to methodical internal treatment for ulcer one should not hesitate to do away by operative interference with the hindrance at the pylorus, which is really the principal cause for the whole trouble. We are more apt to come to this decision when we are certain that we have to deal with ulcer and pyloric obstruction than when we regard gastrosuccorrhœa as a disease *per se*, caused simply by an increased irritation of the mucous membrane. In the literature there are descriptions and post-mortem reports of fatal cases of gastrosuccorrhœa which give one the impression that had the cases been diagnosed as ulcer with pyloric stenosis, and promptly operated upon, the patient could have been saved. In all the cases with Reichmann's symptom-complex which have come under my own observation and which were operated upon, the diagnosis of ulcer was confirmed.

I wish to describe one case in detail because it presents another interesting condition with which gastrosuccorrhœa stands in intimate relation, namely, tetanic attacks.

*History.* In May, 1898, the patient, aged forty-four years, stated that for the past two years he had suffered with heartburn and gastric pain of a dull and at times even intense character, occurring several hours after eating, but particularly at night. In the morning, before eating, he was troubled with a gnawing sensation in the stomach and gaseous distention; no vomiting; bowels regular. The trouble had been regarded as nervous dyspepsia and treated as such.

The patient was tall, thin, had a long thorax with healthy thoracic viscera, a flat abdomen and normal outlines for liver and stomach, the pyloric end being painful to pressure. Urine now and later throughout the whole course of the disease free from albumin and sugar and contained a moderate amount of indican.

Examination after test meal (tea and toast) showed moderate hyperacidity with impaired amylolysis. Alkalies and a suitable diet relieved the subjective symptoms for a few months, during which time patient gained six pounds. In the fall the symptoms returned with increased intensity, particularly the pains during the night.

Stomach analysis of November 19th showed strong hyperacidity. As ulcer had been suspected from the beginning, a treatment for ulcer was suggested, but again refused, as formerly. The patient did not place himself under treatment until the end of January, 1899, when the pain became so intense that it robbed him of sleep.

The first lavage (February 16th) showed a black fluid without food particles in the fasting stomach, so that there could be no more doubt as to the presence of an ulcer. From then on rest in bed. For four days nourishment exclusively by rectum—then in the morning Carlsbader Mühlbrunnen and, besides the enemata, milk per os, at first in small quantities, gradually increasing.

Since three weeks of this treatment gave but little relief from pain, and alkalies, bismuth, belladonna, codeine only relieved the patient temporarily, lavage was taken up and after the washing bismuth was introduced. The fasting stomach nearly always contained from 50 cm. to 100 cm., and occasionally more, of a strongly acid fluid, mostly stained with blood and often mixed with some remnants of food of the previous days.

The bismuth treatment alleviated the pain but little. Irrigations with silver nitrate (1:1000), which were begun two weeks later, also produced little effect. Changes in the milk diet (cooking with fine flour, peptonizing, sour milk) and a trial of thick gruels and soups gave no better results. On the contrary, during the second half of March the pain increased in intensity and duration. Since the rigidly applied internal treatment failed absolutely, operation was advised on March 24th, but had to be postponed several days for ulterior reasons.

During this time there was a decided change for the worse. Until then the quantity of fluid found in the fasting stomach had been relatively small, even when larger quantities had been ingested. During the middle of March, with the increase of pain, the amount of stagnating fluid became greater. From the 24th to the 29th signs of complete pyloric obstruction set in, with almost continual excruciating pains. Although smaller quantities of food were taken the amount drawn from the stomach increased; rapid diminution of urinary secretion, which so far had been plentiful; skin dry; emaciation more marked. Now, for the first time, a hard, painful resistance could be felt in the pyloric region.

*March 25th.* But little nourishment was taken; during the night, after most violent pains, for the first time there was vomiting of blood-stained fluid. In spite of the large quantity of the vomitus, and although nothing had been taken afterward, 750 cm. of fluid were withdrawn from the stomach in the morning of March 26th. During this day the patient took only 180 c.c. each of milk and Vichy. Yet in the evening, when the continual severe pain made it necessary to again introduce the stomach-tube, as much as 1100 c.c. of blood-stained fluid was obtained—that is, three times the quantity taken during the whole day. Upon standing a thin, reddish-film formed on the surface of this fluid, the remainder consisting of clear, light green liquid, with a very high percentage of hydrochloric acid.

During this last introduction of the stomach-tube violent vomiting set in, and with it appeared tonic spasms of arms and legs, both being stiffly extended, the fingers in obstetrician's position. The spasm lasted several minutes. Feeling of numbness in the extremities. Trousseau's phenomenon negative.

*27th.* A total of 600 c.c. of milk was ingested. In the evening a litre of sanguineous fluid of intensely acid reaction was removed. During the withdrawal of this fluid through the tube (this time without vomiting) both upper extremities became tonically extended, the fingers of the left hand were extended, those of the right in the position of a penman. The spasm relaxed slowly several minutes after the washing was finished. Lower extremities not affected this time. Trousseau's phenomenon negative.

*28th.* Condition improved. Pain diminished. Larger quantities of milk taken, and it passed into the intestine. Urinary secretion again reached 1000 c.c.

*29th.* Patient was taken to Dr. Lange's clinic. Lavage of the stomach at 11 A.M. One hour later sudden violent pain in the gastric region and symptoms of collapse. Pulse small; face pale; syncope; abdomen drawn in; liver dulness normal. All the abdominal muscles rigid as boards, several fibres standing out as sharp ridges above the level of the skin. Less pronounced cramps in the muscles of the trunk and extremities. The extremely painful tetanic contraction of all the abdominal muscles remained unchanged for hours, despite the injection of morphine and a mild anæsthesia from ether and chloroform.

Four hours after the perforation laparotomy was performed. The anterior wall of the stomach showed a flat, almost circular swelling, about 5 cm. in diameter, its firm edges extending downward to the greater curvature, and its right limit being still several centimetres removed from the pylorus. The serous covering of the swelling lay free in the peritoneal cavity, only its median edge being adherent to the omentum. Exactly in the centre of the swelling was a sharp-edged opening of the size of a pin-head; there was some turbid fluid in the neighborhood of the perforation.

A segment as broad as a hand was resected, which included the antrum pylori and the pylorus; suture of the stomach end; duodenal end joined to the posterior wall of the stomach by a Murphy button. The resected piece, cut open along the lesser curvature, showed on its inner surface an ulcer as large as a fifty-cent piece, with an uneven terraced surface, a very thin floor, and firm, undermined, wavy edges. Cut surface through this edge showed firm, white lines. Microscopically, adenocarcinoma. The pylorus thickened but patent. Microscopic examination of the pylorus showed greatly hypertrophied muscles, but no signs of carcinoma.

The healing of the wound progressed smoothly. Nourishment at first per rectum. Upon giving food by mouth recurrence of pyrosis and pain in the stomach; perhaps this can be partly accounted for by the retention of the Murphy button. These complaints, which were somewhat relieved by alkalies and washing of the stomach, manifested themselves after each meal, no matter what kind of food had been taken; and they continued to disturb the patient in the most annoying manner during the following months almost up to the time of his death.

An examination after test breakfast on May 25th again revealed considerably increased gastric secretion. Repeated examinations made after this date showed the continuance of hyperacidity for a remarkably long period, in spite of the development of metastatic tumors.

During the first few months following the operation the patient recuperated and gained in weight. In September the pyrosis and pain became worse again; all attempts to influence the secretion medicinally by means of alkalies, bismuth, atropine, etc., were fruitless.

At the end of September signs of decided stagnation set in, with intense pain and vomiting, so that stomach washings were again resorted to and continued until death, on November 19th, at first once a day, then with increasing stenosis, both in the morning and at night. Upon resuming the lavage the tendency to tetanic spasms recurred, and they were more or less pronounced until within a few weeks before death. They generally manifested themselves in extension of the fingers and hands, more rarely of the arms and legs, and lasted at times only half a minute, but occasionally several minutes.

As early as the end of May—two months after the operation—a distinct resistance could be felt at the umbilicus, and four weeks later hard masses were palpable in the abdomen to the right of and below the scar. They grew to be larger than a fist, pushing the abdominal wall forward. On September 23d, when these tumors had already gained very considerable size, the stomach contents still showed a very high percentage of hydrochloric acid. From the middle of October, however, the quantity of this acid decreased rapidly, and lactic acid appeared. From the end of October the large, fermenting residual masses smelt foul, almost fecal.

Epitome. We have to deal here with a case of gastric ulcer which became carcinomatous in a comparatively short time. To be sure, the duration of the ulcer cannot be accurately determined. The symptoms of which the patient complained for several years had been regarded by competent physicians as manifestations of nervous dyspepsia. Cases of gastric ulcer which are diagnosed as nervous dyspepsia are by no means rare, and here we have another instance of it. But here, as in many other cases, it was the persistency of the pain that aroused the suspicion of the ulcer, which suspicion proved perfectly justifiable when lavage of the stomach showed sanguineous contents. Yet this ulcer had certainly existed some time before this proof was obtained, and had already involved the deeper layers of the stomach, for the operation, performed only six weeks later, showed an ulcer involving the whole thickness of the gastric wall, with firm, undermined edges, which had already undergone carcinomatous degeneration.

On looking back over the history of the case, therefore, we will not err if we presume that all the symptoms which the patient showed during the last three years of his life, including those taken for nervous dyspepsia, were caused by this ulcer, since during all that time the pains varied only in intensity, their character remaining the same throughout.

At the operation we found that the ulcer was situated several centimetres from the pylorus; the pylorus itself was patent and free from carcinoma, but showed greatly hypertrophied muscles.

This hypertrophy can be explained as a result of the frequent spasms which evidenced themselves clinically by the severe pains. The spasms occurred particularly during the night. For how long a period they had caused stagnation of the gastric juice, with or without food remains, cannot be determined, since for a long time the patient refused lavage. When we commenced to evacuate the stomach regularly in the morning the patient had already been in bed for weeks, living exclusively on a milk diet. In spite of these favorable circumstances, there were nearly always found in the fasting stomach quantities varying from 50 c.c. to 100 c.c. of very acid fluid, often containing food particles. Later the residual fluid became greater when the increase of pains in duration and intensity indicated longer spasms, and shortly before the perforation took place there set in under almost unceasing pains long-continued spasms, which caused extended periods of pyloric obstruction. It was this protracted period of pylorospasm which brought about the retention of the large quantities of excessively secreted gastric juice.

When cases like the one just described are considered without prejudice there can be no doubt that the main cause for the accumulation of such large quantities of gastric juice is given in the spastic stenosis which prevents the passage of the secretion into the bowels.

The more severe and the longer the duration of the spasm the larger the amount of fluid.

*Tetanic Attacks.* When the spastic stenosis in our case became more pronounced and large quantities of stagnating gastric juice were vomited or removed through the tube, tetanic attacks set in. These recurred in a milder form several months after the operation, when the increasing stenosis at the outlet of the stomach again required lavage. We find the same conditions in nearly all the cases of gastric ulcer with pyloric stenosis in which tetany develops: there is always the evacuation of large quantities of gastric juice, either by vomiting or through the tube. This points to the possibility that the withdrawal of such large quantities of chlorine which are not replaced causes certain changes in the system predisposing to the occurrence of tetanic attacks. We shall discuss this point later on.

In our case, as in many others, it was the lavage which furnished the direct irritation which reflexly brought on the spasm. Remarkable, however, is the severe and long-continued tetanic contraction of the abdominal muscles in connection with the perforation. Contraction of the abdominal muscles is, indeed, a well-known symptom of perforation into the abdominal cavity. In this case, however, where a strong tendency to spasm, perhaps due to the deficiency in chlorine, already existed, the perforation was the exciting cause for an extremely severe tetanic contraction of the entire abdominal wall.

It was Kussmaul who, in his famous article on the treatment of gastric dilatation by means of the stomach-pump, first described the occurrence of muscular spasm with pyloric stenosis. In some of these cases we have to deal with true tetany, which, as is well known, is characterized by increased mechanical and electrical irritability of the nerves and muscles. Other cases only resemble tetany, or else are epileptiform convulsions with loss of consciousness. The cases described by Kussmaul were pyloric stenoses, in which the frequent vomiting of large quantities of fluid led to a high degree of emaciation and decrease in the quantity of urine. He attributed the convulsions to the diminution of water in the organism, which was brought about by the vomiting of such large quantities of fluid and which led, as he reasoned, to the drying out of the nerves and muscles. This theory was not considered entirely satisfactory. It was claimed that there are other diseases of the gastro-intestinal tract in which an abundance of water is lost without the advent of tetany, and that in cases of tetany the introduction of water into the rectum did not prevent the attacks. Other explanations were, therefore, looked for.

Germain Sée believed that the convulsions were caused reflexly by the irritated nerves of the gastric mucosa. This so-called "reflex theory" explains only how the convulsions are brought on, without mentioning the predisposing element. We must remember, however, that the exciting cause, which may be of various characters, is only a secondary element; in order to bring on a tetanic attack a change in the irritability of the nervous system is required and is really the underlying cause of the whole trouble.

This predisposing element finds more consideration in a third hypothesis, which, thoroughly in accord with modern teaching, attributes the origin of the attacks to autointoxication. This theory is mainly advanced by French authorities, who assume that decomposition-products are formed in the stagnating stomach contents, which, when absorbed, act deleteriously upon the nervous system, and in this way lead to attacks.

However, in spite of diligent research, no one has yet been able to demonstrate such poisons, and Friedrich Müller, a supporter of the reflex theory, points out very correctly that tetany appears, not in cases where the fermenting stomach contents are finally absorbed, but only in those in which the stagnating masses are removed from the system either by vomiting or lavage.

Since the removal of great quantities of fluid from the body is actually the only objective finding which is regularly observed in these cases, it remains, after all, the most important point, and if properly interpreted, gives us a better insight into the origin of tetanic attacks than the purely hypothetical autointoxication theory.

Thus, going back to Kussmaul's first explanation, we must say, as this keen observer rightly maintained, that the great loss of fluid by vomiting or lavage brings about the changes in the system which cause the tetanic attacks. We would add, however, that these discharges deprive the system not only of large quantities of fluid, but also of large amounts of chlorides. For in these cases of gastric dilatation complicated with tetany we often find enormously increased gastric secretion. Since, as we have seen in the beginning, the pyloric stenosis prevents the excessively secreted gastric juice from passing into the intestine, it is removed by vomiting or lavage, and thus great quantities of hydrochloric acid become lost from the system instead of being again resorbed.

Bouveret and Devic claim that gastric tetany is observed exclusively in such cases of pyloric stenosis as are accompanied by excessive hypersecretion. This is not absolutely true. Though the instances are very rare, Fleiner, for example, reports cases in which no increase of gastric secretion was found. We will not investigate now the cause of tetany in these particular cases. Suffice it to say that in gastric tetany, as is the case with other spasms, different factors may contribute to the development of the attacks. In the majority of cases, however, it is a fact that large quantities of chlorine are lost from the body by the removal of the excessively secreted gastric juice. This is proved by the fact that in these cases the secretion of chlorine through the urine steadily diminishes, and finally ceases altogether.

To be sure, the disappearance of chlorides from the urine here is not an absolute indicator of the quantities really lost from the body. The organism holds on to its chlorine obstinately. When we limit the supply of chlorine by cutting it out of the food the quantity excreted in the urine becomes less, and finally disappears altogether. At the same time the secretion of hydrochloric acid with the gastric juice will stop, as was shown by A. Cahn in his experiments on dogs. The organism saves its chlorine by checking its secretion. In spite of the diminished secretion, there may still be a great deal left in the system. In gastrosuccorrhœa, however, the conditions are altogether different. Through the constant irritation to the mucosa here, causing the excessive secretion of gastric juice, large quantities of chlorine are constantly withdrawn from the blood, and since they are removed from the body by vomiting or lavage, reabsorption is prevented, so that the body suffers an actual loss of chlorine. At the same time these patients do not take the proper amount of food, and, therefore, not the proper amount of chlorine to make up for the loss. In other words, there is increased excretion of chlorine, and the amount ingested is decreased—a unique pathological condition which must lead to an impoverishment of chlorides in the system. We may well assume that this diminution of chlorine plays some part in the development of tetany.

In looking through the literature I find that Korszynski and Jaworski as early as 1891 already regarded the decrease of chlorine in the tissues as the probable cause of gastric tetany. It is remarkable that this view can nowhere be found quoted in the text-books, in the special articles on gastric tetany by Fleiner, Albu, and others, or in Frankl-Hochwart's monograph "On Tetany," in Nothnagel's series. Yet their theory is of great assistance for the treatment of that peculiar and dangerous condition.

Korszynski and Jaworski, following their theory, recommended the injection of large quantities of normal salt solution, subcutaneously or by rectum, in order to overcome the chlorine starvation in cases of gastric tetany.

Tetanic attacks are of serious import, and often lead quickly to a fatal issue. In the latest summary by Albu of these cases, forty in all, there were thirty-one deaths—*i. e.*, 77½ per cent. mortality. Their presence influences unfavorably the prognosis of an operation, and no operation should be undertaken in these cases before infusing large quantities of salt solution. This was done in our case before and after the operation, and it probably helped to bring about the favorable result, despite the presence of a perforation. To be sure, there were two other factors which favored the case: first, that the operation was performed only a few hours after the perforation; and, again, that the stomach had been washed shortly before the perforation occurred, thus preventing gastric contents from getting into the peritoneal cavity.

Albu claims that therapy offers no remedy which can either check or prevent a recurrence of the tetanic attacks. But, perhaps, as has been said before, it may be possible to end the attack by means of salt infusions. A recurrence can only be prevented by removing the cause. Their cause is, as we have seen above, motor insufficiency and the spastic or organic pyloric stenosis which hinders the passage of the more abundantly secreted hydrochloric acid into the intestines, thus preventing its reabsorption and depriving the system of large amounts of chlorine. In order to remove the cause the treatment should therefore be directed, first of all, against the motor disturbance. Whenever it is impossible to correct the motor insufficiency by internal means, it should be overcome by operation, either by pyloroplasty, gastroenterostomy, or resection, as the individual case may require. That is the rational treatment for gastrosuccorrhœa, as well as for tetany.

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# CHRONIC GASTRITIS DUE TO ALCOHOL.

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In the large cities we are accustomed to see numerous cases of gastric disorders which can be traced directly to excessive and habitual use of alcoholic beverages. These maladies are very common in the low orders of society which make up the mass of hospital patients, and it was from this prominence of these affections that we became interested in them. It is, of course, well recognized that alcohol can produce a chronic gastritis, but the clinical differences of gastritis due to alcohol from gastritis of other causes is not at present so clear. At present we are concerned only with the secretory disturbances of the gastritis of alcohol. The methods of work were those ordinarily used in the routine examination of gastric contents, Ewald's test-breakfast of bread and unsweetened tea being given in the morning and expressed thirty minutes or an hour later. Free HCl was estimated by titration with $\frac{n}{10}$ NaOH, using dimethylamidoazobenzol and Günzborge reagent as an indicator, and phenolphthalein as an indicator in estimating the total acidity. Lactic acid was always tested for, as were the gastric enzymes.

The cases seen divide themselves into two classes: those presenting symptoms of gastritis and those without such symptoms. Of the first class but little need be said. The symptoms are familiar: nausea, with retching, on rising in the morning, and the vomiting, perhaps, of some mucus. These symptoms pass away after a couple